REVIEW

OF

FEVER: *A Study in Morbid and Normal Physiology, by H. C. Wood, A. M., M. D.*

BY

MARY PUTNAM JACOBI, M.D.

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Fever: A Study in Morbid and Normal Physiology.

W. C. Wood, A.M., M.D., Prof. of Diseases of Nervous System in University of Pennsylvania; Member National Academy of Sciences, etc. [Smithsonian Contributions to Knowledge.] Philadelphia: J. B. Lippincott & Co., 1881, pp. 258.

A mere quotation of the principal conclusions arrived at as the result of the 121 experiments recorded and analyzed in this essay, —would quite fail to do justice to the extensive labors involved in their performance. It would also fail to assign a proper place to the researches of the author among those, more or less analogous, of Liebermeister, Leyden, Senator, and others.

The central problem of fever is the relation of heat production and heat retention to it and to each other. It may not be amiss to compare the conclusions arrived at by different authorities in regard to this problem. Thus, "fever is a symptom complex, depending on an alteration in the regulation of heat, by means of which, heat production is increased above the norm, and heat dissipation so disposed (*angeordnet*), that abnormally high bodily temperature results."—Liebermeister.*

"We must *reject* the hypothesis that fever originates in disorder of the nervous centres; that by means of the influence of the nervous system on the systemic functions, the liberation of heat at the surface of the body is controlled or restrained, so that 'by retention' the temperature rises. * * * We are at liberty to adopt the other alternative, that fever originates in the living tissues, that it is from first to last a disorder of protoplasm, and that all the systemic disturbances are secondary. * * * But if we attempt to do this now, we shall at once find ourselves in face of an unsolved physiological problem—that of the normal

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* "Pathologie und Therapie des Fiebers," 1875, p. 359.

relation between temperature and thermogenesis."—Burdon-Sanderson.*

" In fever, the elimination of heat is increased, both with a constant, a rising, or a falling bodily temperature. It follows therefore, without doubt, that heat production is also increased. At the height of the fever, the elimination of heat reaches from one and a half to about double the normal amount. It is at its maximum in the critical stage, with rapid fall of temperature, there being from $2\frac{1}{2}$ to 3 times the normal. The defervescence is accompanied by marked sweat secretion and water evaporation ; while during a rising fever no evaporation can be discovered. In the epicritical stage, the elimination of heat falls below normal." —Leyden.†

" The excess of bodily temperature during fever cannot be explained by the alterations of tissue metamorphosis, as indicated by its final products, urea and carbonic acid. This excess has different causes, among which *with certainty* may be indicated : 1st. The consumption of tension forces (*Spannkräfte*) stored in the healthy organism for the performance of functions. 2d. The accumulation of heat in the body, during the pyrogenetic period preceding the period of fever. Besides these, may perhaps be reckoned as sources for increased heat production, the greater destruction of albumen to urea, and also processes related in some unknown way to the formation of water. (Increased oxidation of hydrogen according to Auerbach.) The higher temperature in fever is determined by a disproportion between the abnormally increased production and the elimination, which is not increased to the same degree."—Senator.‡

" Fever is a nutritive disturbance in which there is an elevation of the bodily temperature, and also an increase of the production of heat by an increase of the chemical movements in the accumulated material of the body ; this increase being sometimes sufficient, sometimes insufficient to compensate for the loss of that heat which is derived directly from the destruction of the surplus food in the body, very little or no food being taken in severe fever. The rise of temperature in fever is, therefore, not dependent altogether upon increased heat production, as in fever there certainly is sometimes less production of heat in the organism than there is at other times when the bodily temperature remains

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* Report of the Medical Officer of the Privy Council, 1875.

† Untersuch. über den Fieber, *Deutsches Archiv*, Bd. v., p. 305.

† Untersuchungen über den Fieberhaften Process. Berlin, 1873.

normal ; also excessive heat production may occur even at the expense of the accumulated materials of the organism without elevation of the bodily temperature."—Wood.*

In this last definition, the only one which the purpose of this review permits us to discuss, it is noticeable that the question of "heat retention," as a factor in fever, is evaded. It may be indirectly referred to in the statement that "the rise of temperature in fever is not *altogether* dependent upon increased heat production :" but neither in the paragraph, nor in the rest of the author's summary, is his opinion on this controverted question formally stated.† In an earlier formularized statement, however (p. 240), the influence of heat retention is much more distinctly recognized.

"The degree of bodily temperature in fever depends, in a greater or less measure, upon a disturbance in the natural play between the functions of heat production and heat dissipation, and is not an accurate measure of the intensity of the increased chemical movements of the tissues."

The author shows, incidentally, in arguing from experiment 114, where the production of animal heat in a pyæmic dog was found to be excessive, 297.7511 calorics hourly as compared with 84.2426 calorics of feeding day, and 60.156 of hunger (apyretic) day, while the bodily temperature remained more than little above normal, that this disturbance between heat production and heat dissipation may assume a different form, and "an excessive heat dissipation may entirely mask an excessive heat production." Thus "the highest development of the fever process may occur when the temperature is lowest" (p. 236).

This is a most important proposition. The physiological experiments contribute largely to explain the clinical phenomena of certain fevers, especially scarlatina, with its low temperatures in malignant cases, as compared with remarkably high temperatures of other cases relatively benign. It is a proposition constantly to be borne in mind in the midst of the present almost overwhelming tendency to measure the severity of all febrile disorders,‡ almost exclusively, by the degree of bodily heat rendered perceptible to the thermometer.§

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* *Loc. cit.*, p. 254.

† Traube, it will be remembered, explained all the symptoms of fever by a tetanus of the small arteries, and, in consequence, a diminished elimination of heat.

‡ Except, perhaps, diphtheria.

§ Senator, having accepted the alterations of temperature in fever as a very sure and convenient guage for the height of the fever, and ordinarily for its

In this connection is to be remembered that Leyden's calori-metrical observations on human beings showed an increased dissipation of heat in all stages of fever except the initial stage. There is an absolute increase, even when, relatively to the heat production, it is insufficient. These observations are principally utilized by the author as affording proof of an increased heat production, and as such they are discussed theoretically by Senator, and after him by Burdon-Sanderson, and experimentally by Dr. Wood. We shall, therefore, refer to them again.

To return for a moment to the central conclusion of the author's summary : We feel inclined to criticize the second clause which states that " the increase (of chemical movements in the accumulated material of the body) is sometimes sufficient, * * * to compensate for the *loss* of that heat which is derived directly from the destruction of the surplus food in the body." As a mere verbal correction we would suggest that the substitution of the words " should be " for " is," would make the meaning plainer. But a more important defect in the definition is that it has the air of implying that the increased chemical movements in fever indicate an effort of the organism to produce its requisite heat, when deprived of the ordinary resource of food combination ; whereas, it is very evident that the exaggerated destruction of store albumen, characteristic of fever, precedes the establishment of fever diet, as it is known, by the increase of urea (Ringer, Senator), to precede the appearance of fever. In the next sentence, Dr. Wood closely follows Burdon-Sanderson in saying that " in fever there is less production of heat in the organism than there is at other times when the bodily temperature remains normal," *e. g.*, when a large amount of food is consumed, but when the excess of heat, thence generated, is dissipated by means of the heat-regulating mechanism of the body in normal operation. This proposition is, we might almost say, necessarily true, but to judge of the effect on the organism of a given production of heat, the comparison must be made between the different amounts produced with the same resources. The significance of a given heat production must be entirely different when effected at the expense of food recently added to the organism, or of the stores and forces of the organism itself.

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severity, proceeds to point out that " a body whose store of albmuen and whose tissues are constantly being destroyed withont repair, must, sooner or later, cease to live, whether it have a normal temperature or not " (*l. c.*, p. 177). And " this destruction cannot be attributable in the main to increased temperature, since this can exist, as in tetanus, without any such result following" (*l. c.*, p. 176).

It remains none the less important to call attention to the fact that, without lesion of the mechanism for dissipating heat, increased heat production cannot produce fever. Dr. Wood has performed a large number of experiments for testing the production and dissipation of heat in dogs subjected to a variety of conditions.

There are two ways of measuring the production of heat : 1st, by calculating the quantity and heat value of food consumed. But in fever experiments, it is desirable to eliminate this source of heat production, and the calorimetrical measurements have been made, therefore, on fasting dogs. Such measurements constitute the second method for estimating heat production. If the temperature of the body remain constant (observes Burdon-Sanderson) the quantity of heat discharged is *identical* with that produced, and the quantity of heat discharged is estimated by measuring the temperature of water to which the animal body has communicated its heat. To understand the process, however, it is important to remember that it is not the temperature itself, but the kilogramme heat units communicated to the water, which are estimated. This quantity is obtained by multiplying the degrees of temperature to which the water may have risen, by its weight in kilogrammes, since a heat unit equals the quantity of heat required to raise one kilogramme of water one degree.

"Calorimetric measurements," observes Liebermeister, "constitute one of the most difficult tasks of physical investigation. The difficulties are somewhat palliated in physiological calorimetry by the large mass of heat units to be operated on."

As is known, Liebermeister employed a cold bath as a calorimeter for experiments on human beings ; measuring the temperature of the water before and after the body had been immersed for a certain time in it. Many criticisims have been addressed to this method, of which the most obvious is that, as Liebermeister himself admits, the contact of cold water with the animal body greatly increases the heat production of the latter.

After the experiments of Hirn, the nearest approach to precise calorimetrical observations on the human body has been made by Leyden. These were partial, that is, one leg only of the fever patient was enclosed in the apparatus. The elimination of heat from the entire limb, was first calculated for each square inch of its surface, then, from this, calculated for the entire body.

Wood's experiments, like those of Senator, were made upon dogs. The apparatus used by the American physiologist is essentially the same as that devised by his German predecessor. But his experiments have been much more numerous (121 as compared with 31 by Senator), and some new problems and calculations introduced into the investigation. On the other hand, the questions concerning tissue metamorphosis in fever, discussed at much length by Senator, are scarcely alluded to by Wood. In only a few of the latter's experiments is the elimination of carbonic acid measured ; in none, the elimination of urea.

Some details of the method of experimentation will not be out of place, as they are essential both to the understanding of the problem, and to the appreciation of the amount of labor involved in the research.

The bath of Liebermeister is replaced by a metallic box with double walls. The animal experimented upon is placed in the box ; the water to which his body is to communicate heat, is contained between the walls, and thus does not come into immediate contact with the body,—one great defect of Liebermeister's bath. The metallic box is set in a larger wooden box, and the space between the two filled in with some non-conducting substance. Thus, the apparatus remains protected from the influence of the external air. Air for the respiration of the animal is drawn through the inner chamber in which he is placed, by means of external openings, one at each extremity of the box. That for ingress of air communicates freely with the atmosphere of the room ; that for egress is connected by a tube with an aspirator, which draws a current of air first through the box, then through a gas meter and around a thermometer.

The heat given off by the animal is conveyed to the water of the calorimeter, to the iron of which it is composed, and to the current of air passing through the box. The quantity of heat (heat units) thus received is ascertained by multiplying together the number of degrees expressing the rise of temperature, the weight, and the specific heat * of the water, the iron, and the air respectively. To this amount must be added the amount of heat involved in the evaporation or condensation of moisture in the air of the box. For this purpose, a sample of the air leaving the box must be examined for moisture, by being passed through

* The specific heat of a substance, it will be remembered, is the *relation* between the amount of heat required to raise the temperature of a substance one degree, and that required to similarly raise the temperature of an equal weight of water.

chloride of calcium tubes, thus introducing a new complication in the apparatus.

Now, as to those unfamiliar with this class of experiments, it is important to emphasize that the result obtained from these different measurements is not yet the *production* of heat during a given time, but only the amount of heat given off or "dissipated" in that time. There is no means of ascertaining heat production by direct experiment ; it is estimated by means of a calculation, whose elements are : 1st. The total amount of heat dissipated, as measured by the heat units given to the calorimeter. 2d. An analysis of this heat quantity into two parts of different origin : one, the heat reserve previously stored up in the body ; the other, the amount produced in the body during the experiment, *i. e.*, coincidently with the heat dissipation. For this analysis is required a 3d element of calculation, namely, the rise or fall of the temperature of the animal body during the time of the experiment. The rectal temperature is taken before and after the experiment ; the difference, of course, gives the rise or fall of heat during the period of observation.

Liebermeister, in a chapter on "The Human Body Considered as a Calorimeter," has pointed out that, if external dissipation of heat were entirely prevented, all the heat produced in an animal organism would go toward raising the temperature of its body. The rise of temperature in degrees, multiplied into the weight and specific heat of the body, would then give the number of heat units produced ; the receiving and producing apparatus becoming then, as it were, identical. This fact has been utilized by Wood, although in a different manner from that suggested by Liebermeister, who proposed to directly ascertain heat production by cutting off heat dissipation, the subject of the experiment being placed in a warm bath. Wood, on the contrary, *calculates* the amount of "heat reserve" (*i. e.*, the amount of heat units stored up in the body and employed in maintaining the temperature of the body at any given time) from elements indicated, namely, the rise or fall of temperature, the weight of the body, and its specific heat.

If the temperature has fallen, it is evident that a portion of the heat dissipated has come from the heat reserve. The loss of the reserve, calculated as above described, is therefore subtracted from the total dissipation. The remainder constitutes the amount of heat units which must have been *produced* in order to cover the

amount dissipated. If, on the contrary, the temperature rise during the experiment, it is evident that enough heat has been produced to cover the heat dissipation, and also to add to the reserve. The numbers indicating the two amounts are therefore added together ; the sum represents the total heat production.

It seems to us that this calculation is really a considerable addition to the researches of Senator on the problems of heat production and heat retention. Although the conclusions enunciated by Senator, as the result partly of his own experiments and partly of those of Leyden on human beings, sound very positive in the summary, and as quoted by Burdon-Sanderson, closer examination shows that their author feels a good deal of uncertainty about them.

" The alterations of heat dissipation," he observes, * * "may be explained without any alteration of heat production; and the fact that the bodily temperature is maintained above the norm is quite compatible with *no* increased heat production, since calorimetric experiments have demonstrated, beyond a doubt, that pyæmic fever begins with a retention of heat. * * * In case heat production is on the whole increased during the fever process, it is certain that in the dog, at least, this increase cannot be great. * * A moderate increase corresponds with alterations of tissue metamorphosis (shown to be quite insufficient to explain the entire rise of temperature). * * * Observations, as on the 2d fever day of the 1st series, on the 3d fever day of the 3d and 7th, when more heat was dissipated than in the corresponding apyretic day—and, nevertheless, the body, at the end of the experiment, became warmer than at the beginning, —justify the opinion that *here* an abnormal amount of heat has been produced. On the other hand, observations in the 2d, 6th, and 7th series, where the animal dissipated abnormally little heat, and yet the temperature of his body fell, should lead to an entirely different conclusion." (Senator, *l. c.*, pp. 85 and 83.)

In Wood's six experiments the heat production and heat dissipation remained constantly parallel, rising and falling together. This appears from the summary of a single experiment, which, in this particular, is a sample of all. The calculation is in heat units.

The bodily temperature, however, is in no fixed proportion to the observed heat dissipation, nor, consequently, to the calculated heat production. In some cases it remained unchanged. Then the production and dissipation of heat were necessarily

equal. Thus (p. 191), " 12th period. No change in bodily tem-
perature. Hourly dissipation of heat=hourly production of heat
126.7412." In other cases it fell, though the dissipation of heat
was less. Thus, " 11th period. Fall of temperature in 1 hour
0.055°, hence heat taken from reserve=1.60875, while hourly
dissipation of heat=117.946, and (calculated) hourly production
=116.337." Sometimes it rose, while the dissipation of heat
remained the same or increased, as, "7th period. Rise of
temperature in 1 hour=0.109°. Heat units added to reserve=
3.04. Hourly dissipation of heat=117.0084. Hourly production,
however (calculated from addition of two),=120.1966."

Day	Time in calorimeter.	Avge. hourly heat dissip.	Avge. hourly heat prod.	Avge. rectal temperature.
Food day . .	17 hours	132.7014	139.4733	104.07°
Hunger day .	18 hours	129.498	128.0702	104.78°
1st fever day .	15 hours	131.5025	130.1177	104.89°
2d fever day .	20 hours	134.243	133.256	105.39°

It is evident that the basis of calculation for these different
elements is essentially the same as in Senator's experiments. In
them increased heat production was positively inferred only when
the bodily temperature rose at the same time that more heat units
were found to have been given off to the calorimeter. Rise of
bodily temperature coinciding with a diminished radiation of heat
to the calorimeter may always be supposed to depend upon heat
retention. It is the fact that, in all stages of fever but the
initial one, heat dissipation has been proved to be increased while
the bodily temperature rises, which constitutes the principal basis
for the theory of increased heat production as an essential part
of the febrile process.

Senator himself observes that his own experiments are inade-
quate for the full solution of the problem, because they do not
extend over a sufficient length of time. There is reason to believe
that both heat production and heat retention are constantly vary-
ing during different periods of the fever, and even of a single
diurnal cycle ; and that the aspect of the morbid process, at any
given time, is the result of a varying combination between the
increase or the diminution of these two factors.* Wood has ex-
tended his observations over fifteen hours instead of three.

* To which Senator adds the variations, observed or inferred, in the tissue
metamorphosis and in other processes, upon which heat production must be
supposed to depend.

In each experiment the animal was examined for twelve different periods, so distributed throughout different portions of the four days that every part of the diurnal cycle could be tested. This extension constitutes an important advance upon the earlier experiments. But it is interesting to notice that the conclusions arrived at do not in any way modify the earlier ones, but are, as the author remarks, " in close accord with those of Senator."

" In the pyæmic fever of dogs, the heat production is usually in excess of the heat production of fasting days, but less than that which can be produced by high feeding ; usually the production of animal heat rises in the febrile state with the temperature and with the stage of the fever, but sometimes the heat production becomes very excessive, although the temperature of the body remains near the normal limit.* In rabbits with pyæmic fever heat production seems to be even greater than it is in health when food is taken " (p. 236).

To the clinician not well acquainted with these recent and most delicate researches in physiology, it may be quite a surprise to learn that excessive temperature and excessive heat production are not identical. It is a most capital fact, however, to bear in mind, if only for its practical bearing on the limits of antipyretic therapeutics.

Before studying the thermic phenomena of fever by means of these experiments on pyæmic dogs, Dr. Wood records a number of experiments performed to investigate the normal mechanism of heat production and heat retention. These are preceded by twenty experiments, showing the effect upon the animal organism of a sudden elevation of its temperature by means of heat communicated from without either to the entire body or to the head. These latter experiments suffice to demonstrate that "external heat applied to the body of normal animals, including man, so as to elevate the internal temperature, produces derangements of the functions of innervation, of respiration, of circulation, etc., etc., precisely similar to those seen in natural fever ; the intensity of the disturbance being directly proportionate to the rise in temperature. * * * The withdrawal of the excess of heat in acute fever is followed by a relief of the nervous and circulatory disturbances " (p. 12). (The latter proposition is demonstrated by two interesting clinical experiments, in addition to those performed on artificially heated animals.)

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* The excessive heat production being masked by an excessive heat dissipation.

" It would appear to follow as a direct corollary to these propositions that excessive temperature is the essential symptom of fever."

Now it seems to us, that in the definition of fever given by the author himself at the conclusion of his essay, and which we have already quoted, the absoluteness of this proposition is very greatly modified. In his summary, Dr. Wood tells us "fever is a *nutritive disturbance* in which there is an elevation of bodily temperature," etc. Here, very properly we think, the essential phenomenon of fever is identified with the "nutritive disturbance." To what extent this may be accompanied by an elevation of bodily temperature, depends, as is abundantly shown by the author in confirmation of earlier experiments, upon quite a variety of circumstances.

The fact that an artificially induced rise of temperature *may* induce derangements of innervation, etc., similar to those occurring in natural fever, by no means proves that in a given case of such fever the other morbid symptoms *are* caused by the bodily heat. For nerve centres, having only limited modes of reaction, may react in the same manner to the direct influence of a poison circulating in the blood. It is only thermic fever (sunstroke) that is exactly imitated by experiments of overheating. The great majority of clinical cases of fever are associated with the introduction into the organism of a specific poison ; and are, therefore, only paralleled experimentally, by the intravenous injections of pyrogenic substances. Dr. Wood recognizes (pp. 12, 13) that in natural fevers "there may be a poison which may modify or entirely suppress the symptoms which the increased temperature would normally produce." But he does not recognize the converse proposition, that this same poison may be the cause of precisely such symptoms as *may* be produced by heat alone. Thus, ataxic delirium (the equivalent in adult human beings for convulsions in the lower animals) may certainly occur in typhoid fever, with only a moderate elevation of temperature. Similarly, the convulsions which sometimes mark the onset of severe scarlatina, may entirely precede any excessive rise of temperature.

Dr. Wood's researches, like those which have preceded them, also suggest an inquiry into the morbid effects of increased heat production when masked by increased heat dissipation, as distinguished from the effects of rise of bodily temperature.

Not only in the nervous and vascular symptoms of fever, but

in the lesions produced during its course, is it necessary to keep in mind the possibility of a double causation,—on the one hand, the high temperature ; on the other hand, the direct action upon the tissues of the original poison. In the practical matter of antipyretic treatment, hardly any two febrile diseases can be compared with each other. Thus, in cerebral rheumatism, the dangerous symptoms do seem to be clearly traceable to sudden heat retention, as indicated and caused by cessation of the profuse sweats that had hitherto kept the bodily temperature within moderate limits, notwithstanding the increase of heat production. Hence, the immediate abstraction of heat is pressingly indicated, and is capable of effecting a rapid and permanent cure. But in prolonged infectious fevers, where the conditions are much more complex, the advantages of the immediate abstraction of heat, by means of cold-water applications, must be compared with possible disadvantages resulting from the increased heat production, which, as Liebermeister has shown, is the *first* effect of these applications.

In studying the mechanism by which the normal animal organism controls the production and dissipation of heat, Dr. Wood discusses experimentally the following problems :

Effects on heat production and heat dissipation of section of the spinal cord. (Exps. 21–24, on rise of temperature. Exps. 25–39, with calorimeter.)

The situation of the chief vaso-motor centre, and the effect on thermogenesis of high section of the medulla. (Exps. 40–53.)

The effect on heat dissipation of paralysis of the vaso-motor centre. (Exps. 54–59.)

The effect on the thermic functions of wounds of the cerebellum. (Exps. 60–62.)

The possible existence above the pons of a heat-regulating centre. (Exps. 72–74.)

The connection between the cerebral cortex and the thermic functions. (Exps. 76–97.)

Possible existence of a muscular vaso-motor centre. (Exps. 98–109.)

Calorimetrical observations, taken, as has been described, before and after section of the cord, showed that this operation was followed by a marked increase of heat dissipation, and coincident lessening of heat production. The author attributes both results to "vaso-motor paralysis, which acts directly (by slackening circulation), and also indirectly, by causing an excessive loss of heat,

and such a lowering of the internal temperature as to check the chemical reactions in the body" (p. 45). The total effect is the marked fall of bodily temperature, which has always been observed after section of the cord.

When section of the medulla was made, through the vaso-motor centre, *i. e.*, just above the point of the calamus, the effect on thermogenesis was the same as that of section of the cord. When, however, the section was made above the vaso-motor centre, *i. e.*, at the junction of the medulla and the pons, not only heat dissipation but heat production was found to increase, the former not keeping pace with the latter, so that the bodily temperature rose. This confirmed the experiments with a similar section, which have been made by Tscheschichin, and also by Bruck and Günter (quoted by author), and which have led to the theory of an "inhibitory heat centre," situated in the brain, in or above the pons. Wood's experiments tend to confirm this theory, and to overthrow that advanced by Heidenhain, namely, that the rise of temperature is due to irritation of the vaso-motor centres. Heat production was found to increase progressively from the time of the section until 23 hours after it ; while, if due to irritation, the heat production should have reached its maximum at once, and then progressively declined. The conclusion is that this heat production was "paralytic, and due to the removal of some active force" (p. 82). It is pointed out that "this theory involves the exercise of a controlling influence of the nervous system upon the nutrition of the body." This remark implies that an excess of heat production always can and must be explained by an increase of the chemical processes, oxidations of the organism. But, as we have seen, either in natural or in artificial fever, induced by other means than medullary sections, the increased oxidations, which can be demonstrated and measured by the elimination of carbonic acid and urea, are insufficient to explain the rise of temperature. We have already quoted the suggestion of Senator, that a portion of fever heat results from the diminution or arrest of work in various portions of the organism, and the coincident liberation of the mechanical heat equivalent of such work. It seems to us that such a mechanical source of heat may be invoked to explain the high temperatures observed after section of the medulla below the pons, with consequent generalized motor muscular paralysis.

The convergence of motor tracts in the pons has been admitted to explain the occurrence of generalized convulsions, when this

part of the brain is irritated, without the hypothesis of a " special convulsive centre." When a section is made between the medulla and the pons, cerebral innervation of voluntary muscles is completely interrupted, and, to that extent, the liberation of motor force prevented. The formation and explosive decomposition of contractile substances in the muscles, however, continue, but the chemical force which is normally converted into both heat and motor force, must now be converted entirely into heat. Hence, we submit a possible cause of rise of temperature, which should increase progressively for some time after the section.

By numerous calorimetrical observations, taken before and after lesions of different portions of the encephalon, it was shown that wounds of the cerebellum produced no perceptible effect upon thermic functions, but that destruction of the first cerebral convolution posterior to the sulcus cruciatus (Hitzig's region) is followed by an increase in heat production ; while irritation of the same nervous tract (salt being placed on the exposed brain) is followed by a decided decrease of heat production. " It seems probable, however (observes our author), that these effects are temporary ; that this first convolution does not contain calorific centres, but is, in some way, connected with these centres."

It is asserted that these centres are probably situated in the pons ; but no experiments are adduced in support of this assertion.

While some influence over heat production was found to be exercised by the cortical centres, Dr. Wood failed to discover any evidence of a vaso-motor action in these same centres. The test of such influence is always galvanization of the sciatic nerve after destruction of the region investigated. If that region contained a vaso-motor centre, irritation of a sensitive nerve after destruction of the region should fail to cause a rise in blood-pressure, since such rise implies the integrity of the vaso-motor centre. The application of this test showed that arterial pressure was quite unaffected by either destruction or irritation of the cortical centres, and this whether the medullary centre were intact, or whether, in order to eliminate its influence, it had been separated by section of the medulla. " Consequently the rise of heat production following section of the medulla is due to an influence not exerted upon the circulation, but directly upon the heat-making function " (p. 157).

This conclusion would not be incompatible with our own interpretation of Tscheschichin's experiment.

In the final chapter of the essay, the author discusses the general theory of fever ; its hæmic or neurotic origin ; the possibility of purely "irritative fever."

In concluding upon these two last questions, Dr. Wood places himself in direct opposition to Burdon-Sanderson, in maintaining that "fever occurring in cases of blood poisoning is often, and probably always, the result of a direct or indirect action of the poison upon the central nervous system, and, hence, is a neurosis. Irritative fever, if it exist, is produced by an action upon the nervous system " (p. 248).

The relations of the vaso-motor system to the febrile state are investigated by comparing the effects of sections of the cord and consequent vaso-motor paralysis in the normal and fevered animals. It was found that, in that latter, the increase of heat dissipation and diminution of heat production, caused by the lesion, were both much exaggerated, over what was observed in a healthy animal. Dr. Wood infers that "the general vaso-motor nerves restrain heat dissipation more completely in fever than in health, and consequently the effect of their sudden palsy is more marked."

This observation is in accord with Senator's on the extremely irritable condition of the vaso-motor nerves in fever, which determines irregular periods of heat retention.

The condition of the so-called inhibitory heat centre in fever was examined in two sets of experiments. In the first, a few hours after the development of fever by subcutaneous injection of pus, galvanism was applied to the femoral nerve, with the effect of causing just such a fall of temperature as has been observed by Heidenhain and others in normal animals. But Heidenhain, using feeble currents, failed to produce this fall of temperature in fever ; hence inferred the paralysis of the inhibitory heat centre.

In a second research the comparative experiments were made on normal and on fever days ; and the irritation was applied to the ear. In this case these irritations were found to have less effect in depressing the temperature than in the normal animal.

We do not think much importance should be attached to these experiments until the mechanism of the thermic functions of the pons be more firmly established. In his summary, Dr. Wood observes : "The only nerve centre proven to exist, capable of influencing heat production without affecting the general circulation, is situated in the pons or above it, and whilst it may be a

muscular vaso-motor centre, it is more probably an 'inhibitory heat centre.'"

"In the pons or above it" leaves a very wide margin, and we have seen that the experiments upon which the above proposition rests are susceptible of a different interpretation.

Still less is there at present sufficient ground for the ninth proposition of the summary : "The so-called inhibitory heat nervous system is not paralyzed in fever, but is less capable than in health of answering promptly and powerfully to suitable stimuli ; in other words, is in a condition of paresis or partial palsy."

The eleventh and final proposition is that "in all cases of serious fever there is a definite poison circulating in the blood, the poison sometimes having been formed in the system, sometimes having entered from without."

It is this depressing poison which acts on the heat-regulating nervous apparatus ; the inhibitory centre is depressed and benumbed ; and, hence, tissue change goes on unrestrained ; more heat is produced ; there is abnormal destruction of tissue. Vasomotor and other (?) heat-dissipation centres are also benumbed, and do not provide for dissipation of animal heat till it becomes excessive.

This last proposition is rather in contradiction with the author's own experiments, showing that vaso-motor palsy is followed by an excessive dissipation of heat.

We have made this long abstract of Dr. Wood's essay because it was needed in order to give any just idea of the range of physiological problems discussed, and of the immense experimental labor which has been devoted to the discussion. From what has been said, it is clear that the value of these important researches consists rather in the confirmation they afford to others already performed, or to theories already distinctly formulated, than to any marked originality in either method or conclusion. The memoir is in no sense "*bahnbrechende*," but its laborious and complex investigations will find on that account no difficulty of acceptance as a solid contribution to the solution of some of the most delicate and abstruse problems in modern physiology. We are not aware that, in this science, any work of similar magnitude has been performed on this side of the Atlantic. It probably could not have been completed without the assistance of the several brilliant collaborators to whom Dr. Wood, in his preface, offers a graceful tribute of thanks. While confirming the principal conclusions of Liebermeister, Leyden, and Senator, its experimental researches

considerably exceed those hitherto performed in multiplicity, range, and variety ; in care and accuracy it yields to none ; and, in some details of calculation, has made a real advance in method. The more familiar the reader may have become with these abstruse researches, the more inclined he will feel to accord high praise to this latest publication of the Smithsonian Institute. [M. P. J.]

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